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Akinetopsia: acute presentation and evidence for persisting defects in motion vision

Akinetopsia—selective loss of motion vision—is rarely described. Current evidence indicates that the brain treats moving stimuli as a distinct feature of vision. Thus, electrophysiological studies have identified cortical areas in the macaque monkey that encode the direction and speed of moving visual stimuli in visual area V5 and adjacent medial superior temporal visual area (MST). Inactivating V5/MST in macaque monkeys induces defects of motion vision that are evident with psychophysical testing or measurement of ocular tracking. Akinetopsia in patients after lesions such as stroke rarely persists, probably because several cortical areas contribute to human perception. Here, we present a patient’s personal account of acute transient akinetopsia following stroke, and report how a patient with transient akinetopsia due a stroke 23 years previously still shows evidence of abnormal motion vision, based on his ocular tracking.

Patient 1 is a 61-year-old woman who sought medical advice the morning after sudden onset of visual disturbance. She had been well, apart from a mild bi-frontal headache until leaving work the preceding day. Travelling home she suddenly noticed that, although static objects appeared normal, smooth movements of people were seen as a series of discontinuous ‘freeze frames’. The opening of a train door was ‘broken up’ and those nearby appeared to ‘move in slow motion’. She was startled when surrounding passengers ‘suddenly moved’. Recognition of people or objects and visual acuity was preserved. These symptoms were unchanged when admitted to hospital 22 h later. She was an ex-smoker of 22 years with an unremarkable past medical history. Her only medication was celecoxib for osteoarthritis. Her account can be found in the accompanying online video.

Her general examination was normal; blood pressure was 167/69. Pursuit and saccadic eye movements were intact on examination but not measured. Colour
vision and visual fields were normal. Her neurological examination was unremarkable. After 2 days her symptoms improved and had resolved by day five. MRI demonstrated tiny multifocal areas of restricted diffusion (hyperintensity on diffusion-weighted imaging (DWI)) and hypointensity on apparent diffusion coefficient map) consistent with recent infarcts in the cortex and subcortical white matter of the inferior parietal lobe and parietal–occipital junction on the right (online supplementary figure 1). Lesions were not detected in her left cerebral hemisphere.

Patient 2 is a 79-year-old former engineer who suffered a left posterior hemispheric stroke at the age of 56, in 1986, causing dyslexia and dyscalculia (see online supplementary data). Soon afterwards, he became aware that objects moving in his right visual hemifield appeared to jump from one location to the next. Thus, when he watched birds flying outside his hospital window and they appeared in his right visual hemifield, they seemed to jump rather than move smoothly. All symptoms improved within a month, although he is still aware of mild difficulties estimating the speed of objects in his right visual hemifield. When studied in 1986, he showed a defect of both saccade and pursuit tracking of targets moving in his right, but not in his left, visual hemifield. This defect was similar to that reported following experimental lesions of extrafoveal V5, and MRI demonstrated a left-sided hemorrhagic infarction affecting Brodmann areas 37 and 19.

When re-evaluated in 2009, uncorrected visual acuity was 20/20 OD and 20/50 OS and he had a homonymous, partial, right superior visual quadrant defect; however, he could easily see the visual stimuli we used. We measured (search coil) ocular tracking of step- and ramp-motion of a small target (measured by abnormal tracking eye movements). Disturbance of motion vision may be detected after symptomatic akinetopsia has resolved, and is not uncommon in those with a history of lesions affecting the cortex and parietal cortex may all contribute to global motion processing. However, the pattern of scattered tiny foci of DWI hyperintensity is consistent with spontaneous fragmentation of thrombus and reperfusion. Without perfusion imaging shortly after the ictus, it remains possible that the initial ischaemic insult was more extensive, or even bilateral. Thus, our MRI findings cannot definitively localize the anatomical substrate for her akinetopsia.

Patient 2 reported disturbance of motion vision restricted to his right visual hemifield, and his left-hemisphere lesion encompassed modern estimates of the human homologue for V5. Twenty-three years later, he reported only minor, persisting disturbance of motion vision, but showed a persisting retinotopic defect of tracking (figure 1) consistent with an extrafoveal V5 lesion. His OFR was more impaired for stimuli presented in his right visual hemifield.

In summary, akinetopsia can present as an acute but transient phenomenon affecting the complete visual field due to unilateral cerebral lesions at more than one site. Long after asymptomatic akinetopsia has resolved, disturbance of motion vision may be detected by abnormal tracking eye movements.

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REFERENCES

Cerebellar-type multiple system atrophy presenting with leucoencephalopathy

In June 2004, a 52-year-old woman was admitted to our department with a walking impairment and scanning speech that had persisted over the previous few months. A neurological examination revealed exclusively cerebellar signs with gait ataxia, slight oculomotor dysfunction and impaired coordination. Known medical and family history were unremarkable up to this point (however, no information could be provided on the patient’s father). MRI scans showed a severe cerebellar atrophy of both hemispheres without any additional pathologies. Electrophysiological (evoked potentials) and blood examination, including vitamin E, vitamin B12, antineuronal antibodies and genetic testing for spinocerebellar ataxia genotypes 1, 2, 3 and 6, were negative for pathological findings. Over the following months the patient developed neurogenic bladder dysfunction with urge incontinence and incomplete bladder release, thus fulfilling the consensus criteria for probable multiple system atrophy of the cerebellar type (MSA-C). We initiated a single photon emission tomography investigation, which revealed a striatal dopaminergic deficit and down-regulation of postsynaptic dopamine receptors; MIIB scans of the heart remained normal. The patient was treated with amantadine up to 500 mg/day without showing any significant improvement. Over time, the disease slowly progressed: the patient needed constant support when walking and she complained of cognitive deficits but there were still no signs of parkinsonism. As the initial MRI scans lacked characteristic findings for MSA-C, we repeated the MRI scans at the end of 2006. As expected, cerebellar...
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